

Case report

Spontaneous spinal epidural hemorrhage following disseminated intravascular coagulation resulting in paraplegia: a case report

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Context: Spontaneous spinal epidural hemorrhage (SSEH) mostly presents as low back pain with or without a radiculopathy, and rarely with paraplegia or tetraplegia depending on the site and severity of spinal cord compression. We present here a case who had anemia and developed paraplegia following disseminated intravascular coagulation (DIC) due to a transfusion reaction.

Findings: A 65-year-old lady presented with sudden onset chest pain radiating to nape of the neck followed by loss of sensations and power in legs few hours after a blood transfusion. Her past history was negative for diabetes mellitus, hypertension, coronary artery disease, or a bleeding disorder. Her blood pressure was 90/57 mmHg and she had a normal pulse, respiratory rate, and temperature. On neurological examination, she had no motor power and unequivocal muscle stretch reflexes in the lower limbs. The sensations were intact till T₃ dermatome. The laboratory evaluation was suggestive of DIC. The magnetic resonance imaging showed a non-enhancing abnormal signal intensity area in the posterior epidural space, extending from CV₄ to LV₄ causing cervico-dorsal cord compression associated with cord edema. Following diagnosis, urgent decompressive surgery was carried out due to deteriorating neurological status. The patient was transfused with five bags of red cell concentrate, two bags of platelets, and four bags of fresh frozen plasma during the operation. The patient regained consciousness following operation, however, the neurological status did not improve. She, unfortunately, died on the third post-op day due to cardiac arrest.

Conclusion: SSEH is a rare cause of paraplegia. Early radiological diagnosis is crucial for timely neurosurgical management and saving patient from permanent neurological deficit or a fatal outcome.

Keywords: disseminated intravascular coagulation, spontaneous spinal epidural hemorrhage, paraplegia

Introduction

Spontaneous spinal epidural hemorrhage (SSEH) is defined as accumulation of blood in the spinal epidural space that has no apparent traumatic etiology. The commonest etiological factors are use of antiplatelet therapy (13–17%), followed by hypertension (12%), rupture of a vascular malformation (9%), hemorrhage into a tumor (6%), and haemophilia.^{1,2} No identifiable etiology can be found in 40–60% of cases.^{3–5} The SSEH mostly

presents as low back pain with or without a radiculopathy, and rarely with paraplegia or tetraplegia depending on the site and severity of spinal cord compression.⁶ We present here a case who had anemia and developed paraplegia following blood transfusion for anemia.

Case report

A 65-year-old lady presented in the accident and emergency department of our hospital around 1:15 am with six hours' history of sudden onset chest pain radiating to nape of the neck while she was sitting in her bed. This was followed by loss of sensations and power in her legs. Two hours earlier than the symptoms onset,

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she had a blood transfusion for anemia. She had a history of prior blood transfusions and was being treated for lumbar spondylolisthesis through analgesics. Her past history was negative for diabetes mellitus, hypertension, coronary artery disease, or a bleeding disorder. On examination, she was conscious and oriented. Her blood pressure was 90/57 mmHg and she had a normal pulse, respiratory rate, and temperature. There were no skin changes, bleeding from mouth, pain abdomen, or breathing difficulty. On neurological examination, she had no motor power and unevokable muscle stretch reflexes in the lower limbs. The sensations were intact till T₃ dermatome.

Her electrocardiogram was normal. The laboratory evaluation showed a hemoglobin of 6.6 g/dL (normal range: 12–15 g/dL), a platelet count of $70 \times 10^9/L$ (normal range: $150\text{--}400 \times 10^9/L$), and a normal mean corpuscular volume, mean corpuscular hemoglobin, and total leucocyte count. Her random plasma glucose was 11.9 mmol/L (normal: 3.3–11.1 mmol/L). The prothrombin time was 20 s (control: 14 s), and activated partial thromboplastin time was 37 s (control: 36 s). Her international normalized ratio was 1.46. The serum fibrinogen levels were 1.18 g/L (normal: 1.5–3.5 g/L) and serum d-dimers were $> 500 < 100 \mu\text{g/L}$ (normal: $< 250 \mu\text{g/L}$) thus fulfilling the laboratory criteria for diagnosis of overt disseminated intravascular coagulation.⁷ Her serum urea and creatinine were raised (16.8 mmol/L and 259 $\mu\text{mol/L}$ respectively) (normal values: 3.3–6.7 mmol/L and 55–105 $\mu\text{mol/L}$ respectively) with a low serum albumin i.e. 28 g/L (normal: 35–50 g/L). Serum alanine transaminase, alkaline phosphatase, and total bilirubin were normal.

The magnetic resonance imaging (MRI) was done on the next day due to technical problems with the machine. It showed a large expansile non-enhancing abnormal signal intensity area in the posterior epidural space, extending from CV₄ to LV₄ causing cervico-dorsal cord compression most marked at CV₄–DV₅ levels. (Figs. 1A and B) There were associated findings of cord edema. Partial collapse of DV₇, DV₁₀, DV₁₁, and DV₁₂ vertebral bodies was seen with abnormal heterogeneous enhancing marrow signals in DV₁₀ vertebral body. (Figs. 2A and B) The later findings were suggestive of a co-existent neoplastic vertebral involvement secondary to metastatic disease or multiple myeloma (No imaging or any other workup was done previously). There were background vertebral degenerative changes. The contrast uptake was not observed thus ruling out other secondary causes of SSEH e.g. arteriovenous malformation, infection, or inflammation.



Figure 1A and B Figures showing a large expansile non-enhancing abnormal signal intensity area in the posterior epidural space, extending from CV₄ to LV₄ causing cervico-dorsal cord compression most marked at CV₄–DV₅ levels along with cord edema.



Figure 2A and B Figures showing partial collapse of DV₇, DV₁₀, DV₁₁, and DV₁₂ vertebral bodies along with abnormal heterogeneous enhancing marrow signals in DV₁₀ vertebral body.

Based on radiological findings, the diagnosis was communicated to the treating neurosurgeon who decided urgent decompressive surgery due to deteriorating neurological status. The epidural hematoma was evacuated following multilevel (CV₅/CV₆, CV₆/CV₇, DV₂/DV₃, DV₈/DV₉, and LV₃/LV₄) decompressive laminectomy. The patient was transfused with five bags of red cell concentrate, two bags of platelets, and four bags of fresh frozen plasma during the operation. The patient regained consciousness following operation, however, the neurological status did not improve. She, unfortunately, died on the third post-op day due to cardiac arrest.

Table 1 Table showing the summary demographics of 39 sampled cases since January 2003

Author	Year	Age	Gender	Level of hemorrhage	Initial neurological recovery score	Primary mode of management	Time since operation in hours	Final neurological recovery score	Comorbidities
Kong ¹⁴	2003	77	Female	C7-T3	A	Surgery	25–48 hrs	A	Hypertension
Kong ¹⁴	2003	52	Female	T8-T10	C	Surgery	13–24 hrs	E	Chronic renal failure, Endometrial Carcinoma
Rosenberg ¹⁵	2003	14	Female	T3-T5	D	Surgery	13–24 hrs	E	-
Cakir ¹⁶	2004	9	Female	T2-T8	B	Surgery	25–48 hrs	E	-
Song ¹⁷	2005	51	Male	T8-L2	A	Surgery	25–48 hrs	A	-
Song ¹⁷	2005	39	Male	T11-L2	A	Surgery	25–48 hrs	A	-
Kelly ¹⁸	2005	31	Female	T2-T4	A	Surgery	< or = 12 hrs	D	Pregnancy
Hsieh ¹⁹	2006	20	Male	C5-C7	B	Surgery	< or = 12 hrs	E	-
SreeHarsha ²⁰	2006	78	Female	T10-L3	A	Conservative	-	E	Hypertension, Diabetes mellitus, Carcinoma Lung, Coagulopathy with heparin use
Sung ²¹	2007	60	Female	T6-T9	A	Surgery	< or = 12 hrs	E	-
Park ²²	2007	68	Female	T7-T12	A	Surgery	< or = 12 hrs	A	-
Akhaddar ²³	2008	47	Male	C2-C7	B	Surgery	< or = 12 hrs	E	Angiolipoma
Oh ²⁴	2008	62	Male	T9-T12	A	Surgery	< or = 12 hrs	E	Hypertension
Finsterer ²⁵	2008	77	Female	T9-L1	A	Surgery	< or = 12 hrs	D	-
Woon ²⁶	2009	42	Male	T2-T4	A	Surgery	> 48 hrs	D	Coagulopathy with warfarin use
Woon ²⁶	2009	33	Female	T2-T4	A	Surgery	13–24 hrs	E	Hypertension
Sirin ²⁷	2010	77	Male	C6-T3	D	Conservative	-	E	Hypertension
Kivity ²⁸	2010	66	Female	T7-T12	A	Surgery	< or = 12 hrs	A	APD, Polycythemia vera
Falavigna ²⁹	2010	76	Male	L3-L4	D	Surgery	13–24 hrs	E	Hypertension, Cerebrovascular accident
Ahn ³⁰	2010	36	Female	C7-T4	C	Surgery	< or = 12 hrs	E	-
Jaeger ³¹	2011	61	Female	C2-T8	A	Conservative	-	E	-
Lim ³²	2011	57	Male	T11-L4	B	Surgery	13–24 hrs	A	Hypertension
Taniguchi ³³	2011	34	Male	T3-T9	B	Surgery	< or = 12 hrs	E	-
Yang ³⁴	2011	56	Male	C2-C4, T7-L1	A	Surgery	13–24 hrs	A	-
Cai ³⁵	2011	35	Male	T3-T4	D	Conservative	-	E	-
Fedor ²	2011	42	Female	T9-T11	A	Surgery	13–24 hrs	A	-
Sasaji ³⁶	2013	37	Female	C6-T6	A	Surgery	13–24 hrs	B	-
Krishnan ³⁷	2014	25	Female	T3-T6	A	Surgery	13–24 hrs	E	Pregnancy
Iwashita ³⁸	2014	60	Female	T1-T9	A	Conservative	-	E	Diabetes mellitus, Hypertension
Giugno ³⁹	2014	75	Female	T6-L3	B	Surgery	13–24 hrs	D	-
Schatlo ⁴⁰	2014	60	Male	T3-L5	B	Surgery	> 48 hrs	E	Chronic liver disease
Kumar ⁴¹	2015	56	Male	T3-L1	A	Surgery	< or = 12 hrs	D	-
Bhat ⁴	2015	68	Female	T12-L2	B	Surgery	< or = 12 hrs	E	Hypertension, Cerebrovascular accident
Coulibaly ⁴²	2015	44	Female	T11-L1	A	Surgery	13–24 hrs	E	-
Alic ⁴³	2016	39	Male	T1-T3	A	Surgery	> 48 hrs	A	-
Ratre ⁴⁴	2016	14	Female	T1-T6	A	Surgery	< or = 12 hrs	E	-
Özyurtlu ⁴⁵	2016	54	Male	T5-T7	A	Surgery	< or = 12 hrs	E	Non-ST elevation myocardial infarction, Hypertension
Aycan ⁴⁶	2016	33	Female	-	A	Surgery	< or = 12 hrs	B	-
Raasck ¹⁰	2017	50	Female	T1-T5	A	Surgery	25–48 hrs	A	-

Discussion

The incidence of SSEH is 0.1 in 100,000 patients per year.⁴ It typically affects people in their fourth or fifth decade of life and men are slightly more affected than women with a ratio of 1.4:1.³ The commonest site of a SSEH is either dorsal or the cervicodorsal spine.⁸ The onset of SSEH typically mimics that of an acute onset polyradiculopathy i.e. sudden pain in the back with radiation along distribution of a nerve root with or without motor deficits in the root distribution. However, rarely, it can evolve to compress spinal cord so severely that the patient develops spinal shock or dies because of its high cervical location.⁸ The other diagnoses that should be differentiated from SSEH are acutely herniated intervertebral disc, transverse myelitis, epidural abscess or tumor, and acute ischemia of the spinal cord.⁹ Contrast enhanced MRI of the whole spine is the primary investigation to assess disease extent and rule out differentials.⁹ Our case had spinal cord compression due to hemorrhage. The vertebral involvement was likely due to previously undiagnosed infiltrative metastatic disease.

The outcome following SSEH has not been studied in relation to etiology so far. The recommended treatment of SSEH is early surgery and evacuation of hematoma.^{3,4,7} The timing of surgery and the prior neurological status are the main factors affecting neurological outcome after surgery.^{4,5,8,10–12} Rapid and extensive deficits and absent sensations are the key components of neurological status affecting recovery.^{10,11} Though, some studies do not support time-bound influence of surgery on clinical outcome,⁹ most link better outcome with surgery done within twelve hours of onset.^{3,4,7,9} The surgery in our case was delayed i.e. was done 16 hours after the onset. Severe anemia and azotemia probably contributed to the fatal outcome.

For cases where patients have a stable neurological status or a contraindication for surgery, conservative treatment with complete bed rest and observation of neurological status can be tried as an option.¹³ The concept behind the conservative management is that SSEH does not clot early and behaves more like a liquid for a longer time thus can travel in the epidural space hence easing compressive symptoms.⁵

In a review of relevant case reports with free full text in the English literature (Table 1),^{2,4,14–46,20} limited to cases since January 2003 with SSEH and paraplegia or paraparesis, ≥ 1 year of age, clear-cut information about method of management, and measurable motor function before and after management, we identified 39 cases with a mean age of 49 ± 19 years (range: 9–78

years). There were 17 (43.6%) male and 22 (56.4%) female. Twenty-four (61.5%) patients presented with neurological recovery score [Frankel's or American Spinal Injury Association Impairment Score (AIS)]⁴⁷ A, eight (20.5%) with B, three (7.7%) with C, and four (10.3%) with D. The commonest comorbidity was hypertension in nine (23.1%) patients. Thirty-three (84.6%) were operated while six (15.4%) were treated conservatively. Fifteen (45.5%) were operated within twelve hours, eleven (33.3%) in 13–24 hours, four (12.1%) in 25–48 hours, and three (9.1%) after 48 hours. Post management, the neurological recovery score was A in nine (23.1%), B in 3 (7.7%), D in 5 (12.8%), and E in 22 (56.4%) patients. Presence of comorbidities and the timing of surgery at ≤ 12 hours or ≤ 24 was not statistically associated with a better neurological outcome (Frankel's or AIS D or E) ($p=0.282$, $p=0.266$, and $p=0.186$ respectively).

Conclusion

SSEH is a rare cause of paraplegia. Early radiological diagnosis is crucial for timely neurosurgical management and saving patient from permanent neurological deficit or a fatal outcome.

Disclaimer statements

Contributors ND conceived the idea, collected data from the patient, and critically revised the manuscript. SBA wrote the initial manuscript, performed the literature search, critically revised the manuscript, and handled the submission process. MOA collected data from the patient and performed the literature search. NA critically revised the manuscript.

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